Central Venous Pressure in Critical Care: The Forgotten Variable

Giles Coverdale, FRCA, FFICM,[1] and Mathew Patteril, MD, FRCA[2]

Central venous catheters (CVC) are used in perioperative and critical care for intravenous access and to allow the delivery of irritant or vasoactive medications. CVCs, when ideally placed, are positioned at or near the junction of the superior vena cava (SVC) and the right atrium (RA) and can be used for hemodynamic monitoring. Central venous pressure (CVP) interpretation has fallen into disrepute in recent years, with high-profile meta-analyses questioning the validity of using CVP to guide fluid administration.[1] International guidelines for sepsis and shock no longer recommend CVP analysis.[2] This article reviews the history of CVP measurement as a hemodynamic monitor, describes controversies in its use, and argues that that CVP interpretation remains a crucial part of hemodynamic monitoring in a critically ill patient.

THE DECLINE IN CVP INTERPRETATION

CVP has been described since the 1950s as a surrogate for RA pressure and by extrapolation as a marker of left ventricular (LV) preload. In "goal directed" hemodynamic management algorithms[3] a "normal" CVP of 8 to 12 mm Hg is commonly an explicit target for volume status. Along with ongoing mixed venous oxygen saturation (SVO_2) measurement, this target was used to guide fluid therapy in a high-profile early goal directed therapy (EGDT) study which led to its proliferation in international guidelines.

From the [1]Department of Anaesthesia and Critical Care, Queen Elizabeth Hospital, University Hospitals Birmingham, Birmingham, UK; and [2]Department of Anaesthesia and Critical Care, University Hospital Coventry and Warwickshire, Coventry, UK.

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Address correspondence to Dr Giles Coverdale, FRCA, FFICM, Department of Anaesthesia and Critical Care, Queen Elizabeth Hospital, University Hospitals Birmingham, Mindelsohn Way, Birmingham B15 2GW, UK. Address e-mail to giles.coverdale@doctors.org.uk.

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However, despite the popularity of EGDT in the 2000s, there were unsuccessful attempts to replicate the benefits of treatment algorithms including a CVP target of 8 to 12 mm Hg in further critical care trials. Studies finding improved outcomes with restrictive fluid strategies opposed the idea that an ideal CVP should be targeted with fluid.[4] Numerous studies have also shown a poor correlation between CVP and a hemodynamic response to fluid. In the last decade, CVP interpretation has fallen out of favor and the 2021 surviving sepsis campaign guideline has removed recommendations to measure CVP, stating that it is no longer advised as a guide to fluid therapy.[2]

RE-INTERPRETING THE DATA FOR "FLUID RESPONSIVENESS"

A high-profile 2013 meta-analysis found poor correlation between CVP values and fluid responsiveness with an area under the curve (AUC) of 0.56.[1] It is often cited as a reason not to use CVP for hemodynamic monitoring. The review included trials that measured CVP and evaluated the hemodynamic response to a fluid challenge. However, the mean CVP for fluid responders in these trials was 8.2 mm Hg compared to 9.5 mm Hg in non-responders with standard deviations of 2.3 and 2.2 mm Hg respectively, suggesting a narrow range of CVP data. The meta-analysis did not test the utility of different cutoff values or the effect of more extreme CVP measurements on fluid responsiveness. This meta-analysis concluded that the use of CVP to guide fluid therapy has no scientific basis and should be abandoned.

Subsequent work has suggested that CVP measurements do have value in predicting response to fluid administration. A 2014 study[5] found that although CVP values between 6 and 15 mm Hg were inconclusive for predicting an improvement in blood pressure following a fluid challenge, values above or below that range showed better correlation. A later meta-analysis[6] acknowledged that most published data was in the 8 to 12 mm Hg range preventing

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generalization of conclusions to CVP values outside this. Despite the paucity of data for extremely high or low CVP values, they found the best thresholds were 2 and 14 mm Hg for fluid responders and non-responders respectively but noted estimates of predictive values were imprecise due to the scarcity of data. At a CVP cutoff of 2 mm Hg the positive predictive value for fluid responsiveness was 65% and at a cutoff of 14 mm Hg the negative predictive value for fluid responsiveness was 66%, both with wide confidence intervals. Taken together, existing literature on CVP and fluid responsiveness do not make a case for abandonment but rather that within a normal range of CVPs, fluid responsiveness may vary among patients, that a shocked patient with a CVP <6 mm Hg may respond to fluid, whereas a patient with a high CVP >14 mm Hg would be unlikely to benefit.

CVP, PERFUSION PRESSURE, AND FLUID TOLERANCE

CVP, which is closely related to RA pressure, provides important hemodynamic information indicating the extent of venous congestion.[7] One way of interpreting CVP, is to acknowledge its role in reflecting venous capacitance, therefore influencing cardiac output and in turn blood pressure. Guyton championed a "venous function curve," which is the relationship of (in essence) CVP vs cardiac output, where the former declines with increasing cardiac output from a peak value at the mean circulatory filling pressure (MCFP). The MCFP is the mean pressure throughout the circulation, measured experimentally (but not clinically) by allowing flow to stop and allowing pressures to reach equilibrium.[8,9] The venous function curve itself is related to cardiac output through the Starling curve (Figure); and in turn these 2 relationships determine arterial pressure, as discussed in detail elsewhere.[10] There is currently no effective way of measuring MCFP in the clinical context. However, Guyton's model suggests that a shocked patient with evidence of end-organ hypoperfusion and an elevated RA pressure (estimated by CVP) would not benefit from further fluid that causes an increase in CVP to supranormal levels.

In addition to this relationship, a high CVP may reduce blood flow to individual organs by reducing the perfusion pressure. Human physiology studies show a drop from a normal arterial pressure to approximately 35 to 45 mm Hg at the end-organ pre-capillary arteriolar vessels.[11] A high CVP would reduce the organ perfusion pressure and flow by diminishing the arterio venous gradient. Adequate lymphatic drainage is dependent on the volume and the pressure gradient, so a high CVP contributes to edema and end-organ dysfunction. A high CVP has been independently associated with poor cerebral saturation and overall poor neurological outcome following cardiac arrest, possibly due to an impairment in cerebral perfusion with increasing CVP.[12]

A high CVP is linked to numerous adverse outcomes including mortality and renal injury in the general ICU[13] and in a range of cardiac disease.[14] The concept of mean perfusion pressure (MPP) has been proposed as the difference between the MAP and CVP. Although no published RCTs have compared an MPP target to a standard MAP target, there is observational data suggesting an independent association between a MPP <60 mm Hg and progression of AKI in the first 12 hours of an ICU admission for sepsis.[15] It is physiologically plausible that an adequate gradient is required between the MAP and CVP to allow

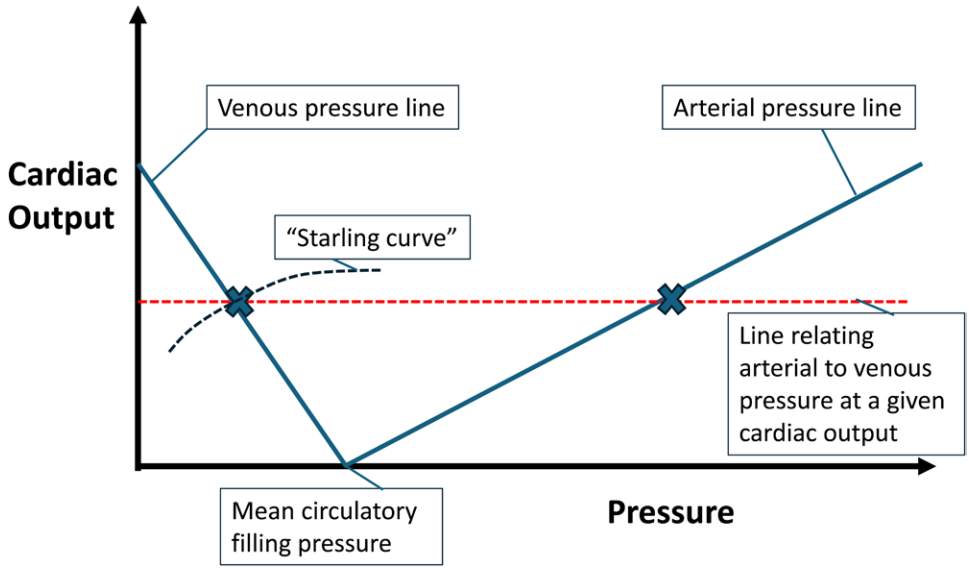

Figure. This graph illustrates the theoretical relationship between arterial and venous pressure for a given cardiac output. The "mean circulatory filling pressure" is where flow is 0 in the system. The gradient of the arterial and venous pressure lines will vary with vascular compliance and cardiac contractility (illustrated by the superimposed starling curve). The figure is adapted with permission from reference [10].

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sufficient organ blood flow. It is therefore reasonable to conclude that a high CVP should signal caution in fluid administration or in any intervention that causes further increases.

Rather than viewing fluid responsiveness and "fluid intolerance" as mutually exclusive, it may be more appropriate to accept that elements of both can coexist to varying degrees. Detrimental venous congestion and a hemodynamic response to a fluid challenge may both be present simultaneously, while evidence of fluid responsiveness alone does not tell us which patients necessarily need more fluid. It is therefore unsurprising that a static CVP value does not correlate well with fluid responsiveness if viewed in isolation. It is more useful to view the CVP as a marker of fluid tolerance with a low CVP providing reassurance that a fluid challenge is less likely to be detrimental when attempting to optimize hemodynamics in shock, than if the CVP is high. Analysis of the CVP waveform, although outside the scope of this article, may also provide important clues to filling status and cardiac function.

CVP AND THE RIGHT VENTRICLE

Consensus statements continue to recommend[16] patients with significant pulmonary hypertension (PH) and RV failure undergo CVP monitoring intraoperatively. Due to its proximity, CVP more convincingly reflects RV diastolic pressures than the LV; a rising CVP in a patient with PH and deteriorating systemic blood pressure is a sign of impending cardiovascular collapse requiring restoration of arterial pressure to avoid RV ischemia and failure. European Society of Cardiology (ESC) 2019 pulmonary embolism guidelines similarly[17] advocate CVP monitoring in acute RV failure. They suggest a fluid challenge to optimize RV preload only if CVP is low combined with a low systemic arterial pressure. A high CVP indicates a risk of acute RV decompensation with fluid.

PH and RV failure is likely to be under recognized in the ICU. It is not restricted to patients with preexisting disease and may develop secondary to the cause of admission, exacerbated by further insults during their stay. The true prevalence of patients in the ICU with PH is hard to quantify but estimates from observational studies range from 28% in the general ICU[18] to 61% in the cardiac ICU population.[19] Observational studies in patients with acute respiratory distress syndrome (ARDS) undergoing lung protective ventilation show echocardiographic evidence of RV dysfunction in approximately 22%.[20] High levels of PEEP, commonly used for lung protective ventilation in respiratory failure, can have detrimental effects on the RV by increasing afterload. This can potentially precipitate dilatation and failure in a struggling RV. A supranormal CVP is an early indicator which should prompt further investigation and measures to preserve RV function; by reducing pulmonary vascular resistance, while optimizing perfusion.

THE ADDED VALUE OF CVP INTERPRETATION

CVCs are frequently placed on the ICU and in high-risk surgery for venous access and drug delivery. There is minimal risk and cost in transducing a preplaced CVC to allow pressure monitoring. The value of de-novo placement of a CVC should be judged on a case-by-case basis, with patients at high risk of cardiovascular instability most likely to benefit, balanced against the well documented risks of insertion and catheter associated bloodstream infection. This article advocates for the use of CVP interpretation in critical illness, where a CVC is also of benefit for drug administration.

Point of care ultrasound and transthoracic echocardiography has become commonplace in critical care and anesthesia over the last 10 years. There are a variety of methods of assessing the consequences of venous congestion and the presence of RV pathology, also providing estimates of intracardiac and venous pressures. These estimates are of variable accuracy in different patient populations and are often user dependent. For example, inferior vena cava (IVC) measurement and collapsibility/distensibility by ultrasound shows generally good correlation with CVP but has significant limitations in mechanically ventilated patients; problematic in anesthesia and critical care.[21] Ultrasound has a subjective element to its interpretation, and a reliance on the availability of trained staff. The CVP however, is a direct pressure measurement of the juxta cardiac great veins (reflecting the RA depending on position) providing a continuous measure of venous congestion. Ultrasound does not effectively replace this but is helpful in conjunction. CVP is a continuous monitor of fluid tolerance and incorporating CVP measurement into hemodynamic assessment may be beneficial in reducing the risk of detrimental fluid administration. A high or rapidly rising CVP, can be an early sign of RV compromise prompting further investigation, another useful tool in a complete assessment of a critically ill patient.

CONCLUSION

There are many arguments that extreme CVP values may still be helpful in guiding fluid therapy. There is now good evidence that a high CVP, regardless of cause, is associated with adverse outcomes. Any interventions that lead to a rapid rise in CVP to supranormal levels should only proceed with caution. A low CVP in a shocked patient is reassuring that a fluid challenge may be beneficial and tolerated. Pulmonary hypertension and RV failure is likely to be an underrecognized phenomenon in the general ICU; CVP can provide a rapid bedside signal for a struggling RV

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that would be intolerant to fluid, prompting further investigation and measures to preserve RV function. This article briefly discusses a possible future where CVP is incorporated into personalized blood pressure targets to allow the preservation of MPP and an appropriate end-organ blood flow. This is an area that requires further research. ■

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REFERENCES

1. Marik PE, Cavallazzi R. Does the central venous pressure predict fluid responsiveness? An updated meta-analysis and a plea for some common sense. *Crit Care Med*. 2013;41:1774–1781.
2. Evans L, Rhodes A, Alhazzani W, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. *Intensive Care Med*. 2021;47:1181–1247.
3. Rivers E, Nguyen B, Havstad S, et al; Early Goal-Directed Therapy Collaborative Group. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345:1368–1377.
4. Wiedemann HP, Wheeler AP, Bernard GR, et al; National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network. Comparison of two fluid-management strategies in acute lung injury. *N Engl J Med*. 2006;354:2564–2575.
5. Biais M, Ehrmann S, Mari A, et al. Clinical relevance of pulse pressure variations for predicting fluid responsiveness in mechanically ventilated intensive care unit patients: the grey zone approach. *Crit Care*. 2014;18:587.
6. Eskesen TG, Wetterslev M, Perner A. Systematic review including re-analyses of 1148 individual data sets of central venous pressure as a predictor of fluid responsiveness. *Intensive Care Med*. 2016;42:324–332.
7. Berlin DA, Bakker J. Starling curves and central venous pressure. *Crit Care*. 2015;19:55.
8. Henderson WR, Griesdale DE, Walley KR, Sheel AW. Clinical review: Guyton—the role of mean circulatory filling pressure and right atrial pressure in controlling cardiac output. *Crit Care*. 2010;14:243.
9. Persichini R, Lai C, Teboul JL, Adda I, Guérin L, Monnet X. Venous return and mean systemic filling pressure: physiology and clinical applications. *Crit Care*. 2022;26:150.
10. Dorrington KL, Pandit JJ. The obligatory role of the kidney in long-term arterial blood pressure control: extending Guyton's model of the circulation. *Anaesthesia*. 2009;64:1218–1228.
11. Levick JR, Michel CC. Microvascular fluid exchange and the revised Starling principle. *Cardiovasc Res*. 2010;87:198–210.
12. Ameloot K, Genbrugge C, Meex I, et al. Is venous congestion associated with reduced cerebral oxygenation and worse neurological outcome after cardiac arrest? *Crit Care*. 2016;20:146.
13. Huang A, Liao L, Pan L, Pinhu L. Association between the central venous pressure and all-cause mortality in critically ill patients with acute kidney injury. *Int J Gen Med*. 2021;14:8019–8027.
14. Damman K, van Deursen VM, Navis G, Voors AA, van Veldhuisen DJ, Hillege HL. Increased central venous pressure is associated with impaired renal function and mortality in a broad spectrum of patients with cardiovascular disease. *J Am Coll Cardiol*. 2009;53:582–588.
15. Ostermann M, Hall A, Crichton S. Low mean perfusion pressure is a risk factor for progression of acute kidney injury in critically ill patients: a retrospective analysis. *BMC Nephrol*. 2017;18:151.
16. Price LC, Martinez G, Brame A, et al. Perioperative management of patients with pulmonary hypertension undergoing non-cardiothoracic, non-obstetric surgery: a systematic review and expert consensus statement. *Br J Anaesth*. 2021;126:774–790.
17. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). *Eur Heart J*. 2020;41:543–603.
18. Stamm JA, McVerry BJ, Mathier MA, Donahoe MP, Saul MI, Gladwin MT. Doppler-defined pulmonary hypertension in medical intensive care unit patients: retrospective investigation of risk factors and impact on mortality. *Pulm Circ*. 2011;1:95–102.
19. Jentzer JC, Wiley BM, Reddy YNV, Barnett C, Borlaug BA, Solomon MA. Epidemiology and outcomes of pulmonary hypertension in the cardiac intensive care unit. *Eur Heart J Acute Cardiovasc Care*. 2022;11:230–241.
20. Mekontso Dessap A, Boissier F, Charron C, et al. Acute cor pulmonale during protective ventilation for acute respiratory distress syndrome: prevalence, predictors, and clinical impact. *Intensive Care Med*. 2016;42:862–870.
21. Ciozda W, Kedan I, Kehl DW, Zimmer R, Khandwalla R, Kimchi A. The efficacy of sonographic measurement of inferior vena cava diameter as an estimate of central venous pressure. *Cardiovasc Ultrasound*. 2016;14:33.

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